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Hazard Characteristics of Combustion Products in Fires: The State-of-the-Art Review

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Center for Fire Research
Institute for Applied Technology
National Bureau of Standards
Washington, D.C. 20234

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Final Report

Sponsored by:
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HAZARD CHARACTERISTICS OF COMBUSTION PRODUCTS IN FIRES: THE STATE-OF-THE-ART REVIEW

Merritt M. Birky

Abstract

The purpose of this effort is to review what is known about the "smoke inhalation" hazard as related to human fatalities, the limitations of the fire fatality data and the methods that have been and are being used to assess the inhalation toxicity hazard.

Fire statistics indicate that 70 to 80% of the fire fatalities are attributed to smoke inhalation. In depth autopsy studies of some of these fatalities show that carbon monoxide is the predominant toxicant produced from fires. The role of new synthetic polymers and other additives is unknown as is the role of hydrogen cyanide in fire fatalities.

Chemical analysis of combustion products has been used extensively to assess the toxicological hazard in fire research. The limitations of such measurements are addressed and a combination of toxicology (animal exposures) and analytical chemistry is recommended. Recent combined biological and selected analytical measurements are critically reviewed.

The mechanism of toxic action of a few well known combustion products is discussed. Due to the extensive use of organophosphates as fire retardants in polymeric materials, the toxicity of this class of compounds is reviewed in some detail.

The role of building codes and standards and early detection and suppression of fire are discussed as a means of reducing human exposure to toxic combustion products. For example, the 1976 French regulation that limits the use of flammable materials that contain chlorine and nitrogen in their molecular structure is presented.

Key words: Biological assessment; chemical analysis; combustion products; fire fatalities; smoke inhalation; toxicity.

1. INTRODUCTION

1.1. Clinical Definition of Problem

Statement of problem: Approximately 80% of the fire fatalities reportedly result from "smoke inhalation."[1] What does this mean?

[1] In the fire literature the term "smoke inhalation" usually refers to the inhalation of the undifferentiated air-borne products of combustion, including gases, vapors, liquid droplets, and solid particles. More precise identification of the causative agent is rarely made. In this report we consider the hazard characteristics of all forms of combustion products.

The U.S. National Commission on Fire Prevention and Control reported that the United States holds the dubious honor of being the leader of all major industrialized countries in per capita deaths and property loss from fire [1][2]. The deaths-per-million population rate in the United States is nearly twice that of second-ranking Canada (57.1 vs 29.7). This leaves little doubt that the United States leads the other industrialized nations even though different reporting procedures make international comparisons imprecise.

A comparison of 1974 international fire losses for various nations was assembled by the National Fire Prevention Association (NFPA) [2]. The NFPA [3] breakdown of these fire losses for U.S. shows that 62% of the deaths occurred in residential occupancies. Bowes [4] reported similar findings for the United Kingdom.

In a further breakdown of fire statistics, Clarke and Ottoson [5] surveyed NFPA fire data to identify and rank what appear to be the most frequent scenarios for fire death in the United States. They found that the most common fire death scenario is the residential furnishings fire caused by smoking materials. This scenario was estimated to account for 27% of the fire deaths.

Bowes [4] prepared a survey of the United Kingdom fire casualties based on reports of the fire brigades for the period 1955 to 1971. He concluded that the proportion of fatalities attributed to smoke and gas during this period increased by a factor of about three (3) while the number of fatal casualties from all causes in fires showed only a small upward trend.

According to Thomas [6] approximately 80% of the victims of fire are not touched by flames, but die as a result of "smoke inhalation." Smoke inhalation represents one of the major hazards of fire, both to civilian and to firefighters. Two detailed autopsy studies of fire fatalities [7,8] indicate the nature of the smoke inhalation problem.

First, Zikria's [7] analysis of autopsies performed on fire victims in New York City during 1966 and 1967 confirm Thomas' conclusions. Zikria found that of the 311 victims that were autopsied, 185 survived less than 12 hours. Carbon monoxide poisoning was the probable primary cause of death [carboxyhemoglobin (COHb) greater than 50%)] in 24.3% of these fatalities. In another 34.5%, CO was believed to be a significant factor. In 11.4%, or 21 cases, the COHb level was 10% or less and in the remainder of the cases [55] carboxyhemoglobin measurements were not made. Very little data was obtained on COHb levels in the victims that lived longer than 12 hours.

In the second study by the Johns Hopkins University, detailed autopsies were performed on 129 Maryland fire fatalities for the period October 1971 to January 1974. In this study reported by Halpin et al. [8], blood carboxyhemoglobin and blood alcohol measurements and a careful dissection of the coronary artery were performed in all cases. Analysis of the results indicated the following: a) carbon monoxide was the primary cause of death in 50% of the cases and b) in another 30% of the cases carbon monoxide plus the presence of pre-existing heart disease, alcohol and burns (including respiratory) were contributing factors. Approximately 10% of the fatalities were due to burns and 10% remain unexplained. The role of other toxicants, including HCN, in the above fatalities has not been established (368 autopsies have been performed to data with the same findings).

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[2] Numbers in brackets refer to the literature references listed at the end of this paper.

A third fire fatality autopsy study has been initiated by the Fire Research Station in England [9]. This study is being done at the University of Glasgow, Scotland and should give additional information as to the nature of inhalation toxicity resulting from fire exposure.

The role of particulates in causing casualties has not been adequately defined in past studies. Obscuration of vision which interferes with escape and hinders firefighting and rescue operations, is generally believed to be a significant factor, but no quantitative measure can be placed on its effect. Indeed, the victim who fails to escape due to obscuration of vision will appear in the record as a burn or toxic gas casualty and there is no way of knowing whether they would have survived had good visibility been maintained.

Particulate matter may also provide a means of transport and deposition of toxic products into the respiratory tract [10-12]. In the past such effects have seldom been differentiated from those of toxic gas inhalation. The fire casualty study at the Johns Hopkins University is now beginning to investigate this problem [8]. In addition to acute toxicity effects, chronic effects due to deposition of heavy metals and other unidentified toxicants may be significant, particularly in the case of firefighters and others subjected to repeated exposures.

Despite the lack of definitive information on the importance of particulates, a considerable effort is being devoted to the development of methods of measuring particulate formation, the characterization of particulates, and the development of materials with decreased potential for obscuration of vision. A few standards which set limits on the smoke producing properties of materials have been adopted or proposed (see section 2.2.). It would be highly desirable to be able to relate these efforts to the hazard they seek to address in a quantitative manner.

1.2. Limitations of Clinical Data

Statement of problem: Is the inhalation hazard of past fires predictive of future problems?

The above clinical data illustrate the nature of the inhalation toxicity hazard associated with the fire environment. Carbon monoxide appears as the single major cause of death in the above fatalities. However, the limitations of these findings should be recognized. In the Hopkins study, on-the-scene fire investigations are carried out to determine the type of materials involved in the fire in order to determine if certain materials present an unusual toxicological hazard. To date most of the fires involved rather conventional materials. That is, there does not appear to be a significant involvement of new synthetic materials. As a result the hazard of new materials remains unknown. The retrospective nature of these data must be recognized and as more and newer synthetic materials are introduced into commerce the nature of the hazard may change. Past problems may not be future problems.

The significance of hydrogen cyanide and other toxicants besides CO in fire fatalities and casualties is still unknown. Similarly the effects of sub-lethal concentrations of toxic combustion products, especially on those exposed repeatedly such as firefighters, have not been delineated.

1.3. Role of Synthetic Materials and Fire Retardants

Statement of problem: Is the technological fix for flammability creating a worse toxicological hazard?

It is frequently suggested that the new materials being introduced into the built environment have the potential for creating an unrecognized toxicological hazard. Some new synthetic materials tend to increase the amount of smoke during a fire and the potential of changing the toxic gas hazard exists. At the present time, there are no statistics to prove that the toxicity hazard is worse as a result of the use of synthetic materials. However, it is recognized that many synthetic materials ignite more readily and the surface flame spread rate may be greater than for some of the natural materials.

In an attempt to reduce the fire hazard, mandatory standards have been instituted to reduce the ignitability and flame spread rates of certain materials depending on the end use. One of the ways technology has responded to this demand is by the incorporation of fire retardant additives [13]. The effect of these retardants on the toxicity of the combustion products is now becoming a cause for concern, due in part to the type of compounds that are being added: i.e., organic compounds containing phosphorus and halogens. The toxicity of organophosphate compounds with and without halogens, such as chlorine and bromine, will be discussed in section 4. At the University of Utah [14,15] a toxic bicyclic phosphorus containing ester has been identified as a combustion product from a fire retarded rigid polyurethane foam based on a low molecular weight propoxylated trimethylolpropane. This example serves only to illustrate the concern caused by the use of fire retardants based on organophosphorus compounds. However, this single finding should not be used to indict all fire retarded polymeric materials.

Some of the additives that are proposed for fire retardants may be inherently toxic even when a fire does not occur. For example, a recent article by Bellett and Casida [16] on the high toxicity of bicyclic phosphorus esters should be noted since the authors suggest that these compounds are candidates for flame retardants, vinyl stabilizers and antioxidants and thus may find their way into polymeric materials. A perchlorinated, cage-structured hydrocarbon ($C_{10}Cl_{12}$) has been identified as an environmental pollutant and the author, Kaiser, [17] states this compound has been promoted as a flame retardant additive for polymers. Similarly, the brominated biphenyls are being used as fire retardants [18]. Their structural similarity to the restricted chlorinated biphenyl suggests that similar environmental and toxicity problems may exist and raises some serious questions about the advisability of such usage.

It should however by recognized that the use of fire retardants based on today's technology has the potential for creating two rather distinct toxicological problems. The first is the toxicity of the fire retardant (FR) itself, e.g., without thermal modification of the material. Exposure may result from normal usage by leaching out, or accidental loss of the fire retardant or during disposal. This type of exposure is likely to be a long-term chronic exposure, perhaps by way of contamination of the environment. Leaching out of the monomer and plasticizers from polymeric materials, especially food and drink containers, may also create a similar toxicological problem. The second toxicological problem, which is exemplified by the bicyclic phosphate ester, occurs only during a fire. This exposure is likely to be a single acute dose, except perhaps for the firefighter in which case repeated acute exposures could occur. Depending on the biological activity of the toxicants, the toxicological effects of the two different types of exposure will be quite different.

A symposium sponsored by the National Academy of Sciences and the University of Utah [19] on the toxicology of products produced from the thermal decomposition of polymeric materials indicates the world-wide concern with combustion product toxicity.

2. METHODOLOGY FOR ASSESSMENT OF SMOKE AND TOXICOLOGICAL HAZARD IN FIRE

Statement of problem: How does one realistically assess these hazards? Can small scale laboratory measurements be made? If so, what are these measurements?

2.1. Background

Except for limited autopsies of fire victims discussed in section 1, the world effort in the past to assess the toxicological hazard associated with fire has been confined to laboratory measurements that fall into three broad categories:

1. analytical assessment
2. biological or bioassay assessment
3. combined analytical and biological assessment.

Of the three categories, major emphasis has been placed in the first category. This includes smoke obscuration measurements and chemical analyses for specific toxicants in small scale laboratory experiments and in large scale fire tests. The analytical measurements are usually further divided into two major types of determinations: the particulate fraction (smoke) and gaseous fraction. The separation of the smoke (particulates and aerosols) measurement from the gases is usually done by necessity due to the fact that these two components have quite different properties that require different measurement techniques for their quantification.

2.2. Measurement of Particulate Combustion Products

Standard methods for the measurement of the concentration of particulates in combustion products are further advanced than are methods for measuring toxicity. This is undoubtedly due to the greater ease of making measurements of the first type rather than to their relative importance. It has generally been assumed that toxic gas formation will parallel smoke production, and thus control of the latter will provide a measure of control over the former. This assumption has been rationalized since both are products of incomplete combustion and the conditions which favor incomplete combustion, low temperatures and limited oxygen supply, should favor the formation of both particulates and toxic gaseous products. Recently, King [20] has examined this assumption experimentally. He found that smoke formation was accompanied by carbon monoxide formation but he was unable to establish any quantitative correlation between the two. His work illustrates the well-known fact that the production of both CO and particulates is strongly dependent on oxygen concentration, incident energy flux to the sample, and other experimental variables. One concludes that there is no established relationship between a smoke standard or measurement and the toxicity problem.

Two basic techniques have been used for the measurement of particulates in combustion products: the optical measurement of light attenuation in the gaseous suspension, and collection by filtration or impingement and subsequent gravimetric or optical measurement. Since obscuration of vision has been considered to be the principal hazard, the optical attenuation methods have been favored for product development and regulatory purposes. If the transport and deposition of toxic agents by particulates proves to be a serious problem, collection methods may prove useful. They have the advantage of providing samples for analysis and physical characterization.

Smoke test methods have been reviewed recently by an ASTM Task Group [21]. Three standard test methods have been used extensively in the United States for the measurement of the smoke producing potential of materials for regulatory purposes. These are the ASTM E 84 (Tunnel) Test, the ASTM D 2843 (XP-2 chamber) Test, and the NFPA 258 (Smoke Density Chamber) Test. Other methods are used in various laboratories for research and development purposes, but they have not been developed to the point where they are suitable for regulatory purposes.

The E 84 method is applicable to wall and ceiling finishing materials. It has also been used for floor coverings although this usage is not covered by the standard. The sample is burned on the tunnel ceiling under a single set of exposure conditions in a well ventilated, intense gas flame. The percent light obscuration is measured in the exhaust stack over a period of ten minutes. This allows some estimate of the rate of smoke formation as a function of time, but apparently no attenpt has been made to use this feature of the test for regulatory purposes. Instead, the percent light attenuation is integrated over the 10-minute test period and this figure is compared to that obtained when a red oak standard is burned under similar conditions. Since the light attenuation is not a linear function of the quantity of smoke, it is not possible to attach any quantitative physical meaning to the value of the integral and there is no way to scale the results to larger fires.

In some cases the rate of smoke production is still increasing at the end of the 10-minute measurement period. The reproducibility of smoke measurements by the E-84 is less than is desirable for a standard test method [22].

The D2843 test method is applicable to the measurement of the smoke producing properties of plastics. The sample is burned under strong heat exposure conditions with limited ventilation. The percent light attenuation in the test chamber is measured by means of a horizontal photometer located about two-thirds of the height of the chamber from the floor. Since smoke mixing takes place through thermal convection considerable layering occurs and the smoke density at an arbitrary height is of questionable significance. The maximum smoke density is recorded as percent light absorption and the light absorption curve is integrated over a four-minute time interval to give a "smoke density rating." Again, the percent light absorption is not a measure of the amount of smoke produced and since the smoke accumulates in the chamber during the period of the test the practice of integrating the absorption curve over a time interval has even less justification than in the case of the E 84 test method.

The NFPA 258 test method measures the total amount of smoke released from a material in terms of the specific optical density, D_s, defined as

$$D_s = \frac{V}{AL} \log F_o/F$$

where V = volume of test chamber

A = surface area of specimen

L = length of light path

F_o = incident light flux

F = transmitted light flux.

The specific optical density is a non-dimensional quantity that provides a quantitative measure of smoke formation. By applying appropriate dimensional relationships, the optical density to be expected under the conditions of a specific fire situation can be estimated.

The smoke density chamber has the photometer light beam oriented vertically, so it measures an average optical density through any layering of the smoke. The sample is oriented vertically and exposed on one surface (wall position). Some thermoplastic materials melt and flow out of the sample holder, producing erratic results. A recently developed modification using a horizontal sample holder should help to correct this problem [23].

The smoke density chamber method recognizes that the smoke producing properties of materials may depend strongly on the conditions under which they are burned. While no test method can simulate the entire range of conditions that may be encountered in real fires, the method measures smoke formation under two different well-defined sets of conditions representative of typical fire environments. Thus it provides a better indication of material performance than measurements made under a single set of conditions. The sample is exposed to a radiant energy flux of 2.5 W/cm^2. Measurements are made in the absence of open flaming (smoldering combustion) or in the presence of a pilot flame (flaming combustion). Materials may give quite different results under these different conditions. Cellulosic products tend to produce more smoke under conditions of smoldering combustion while many synthetics produce more smoke during flaming combustion [24].

2.3. Analysis of Gaseous Products

A review of the literature suggests that the efforts to assess the toxicological hazard in the fire environment by analytical methods can be divided into two types of measurements. The first is a detailed analysis of the products of pyrolysis, and in some cases combustion, using such methods as gas chromatography and mass spectrometry. The second type of measurement assumes the hazard is due to a few selected gases and analyzes only for these products. These measurements are limited to the gaseous fraction with the smoke (particulates) being treated separately.

2.3.1. Detailed Chemical Analysis of Gases

Early efforts to identify the products of thermal degradation were not tailored toward toxic product assessment. The work of Madorsky [25], for example, was designed to elucidate the mechanism and kinetics of polymer

degradation. Nonetheless, product identification as a function of temperature was an important aspect of his efforts. A wide range of polymers was studied including cellulosics, polyolefins, fluorocarbons, sytrene, acrylates, natural rubber, polyoxides, polyamides, etc.

Woolley et al. [26] analyzed the degradation products from flexible polyester and polyether polyurethane foams based on toluene diisocyanate (TDI). The degradation products of the yellow smoke that is produced from urethane were also studied. The experiments were carried out in nitrogen, and the product identification was done with a gas chromatograph and mass spectrometer. At least 30 products were separated on one chromatographic column from the polyester smoke produced at 850 °C. Subsequent efforts by Woolley and Wadley [27] showed that TDI is also produced during the degradation.

Hileman et al. [28] analyzed the products resulting from the thermal degradation of a commercial flexible polyurethane based on TDI. They confirmed that TDI is produced during the thermal degradation of the material and suggested a mechanism for the degradation. Numerous products were identified.

Similar efforts to analyze the degradation products of polyvinylchloride (PVC) have identified a large number of products. An extensive review of the literature on the pyrolysis and combustion of PVC and the effects of plasticizers, stabilizers, etc., on the decomposition products was given by Wagner [29]. O'Mara [30] analyzed the products from PVC resin and plastisols.

Polyvinylchloride was studied extensively at Boreham Wood by Woolley [31] to assess the "real-life" fire hazard from this material. He examined the pyrolysis products from a commercial rigid PVC decomposed in nitrogen and in air between 300 °C and 600 °C. Approximately 75 organic materials were separated by gas chromatography and identified by mass spectrometry. Special care was taken to analyze for phosgene and vinyl chloride. Vinyl chloride was detected and quantified. No phosgene was found at the detection limit of the instrumentation (50 ppm). An evaluation of the toxicity of the products from PVC was obtained by Woolley [31] based on the toxicity index introduced by Tsuchiya and Sumi [32]. He concluded that the main toxic risks from the decomposition products of PVC are hydrogen chloride and carbon monoxide. This index will be discussed in section 2.4.

The pyrolysis gas from an impressive list of polymer systems, including PVC samples, has been analyzed both qualitatively and quantitatively by Boettner et al. [33]. Some 50 compounds have been identified qualitatively from PVC. In addition to PVC, Boettner et al. [33] studied polysulfone, polyurethanes, polyimides, phenol formaldehyde, urea formaldehyde, polystyrene, polyethylene, polypropylene, polyester, polycarbonate, polyphenylene oxide, synthetic fabrics and certain natural materials such as wood and wool.

2.3.2. Selected Gas Analysis — Small Scale

Statement of problem: Can chemical analysis of selected gases in small scale laboratory tests determine the nature of toxicity of combustion products?

The second approach of assuming what toxic species are produced and only analyzing for these products has been used extensively in the assessment of toxic risk due to fire. This philosophy was used in the extensive work reported by Gross et al. [34] involving aircraft interior materials. In this study 141 materials were evaluated in the NBS smoke chamber for smoke production, quantities of CO, HCl, HCN, and in some cases NO_x, HF, SO_2, NH_3, $COCl_2$ depending on the polymer being decomposed. The gases were measured mainly with gas detector tubes and smoke was measured by optical attenuation.

The data accumulated by Gross et al. were presented in a comparative fashion, with various material types such as carpets, fabrics, sheets (panels), laminates, and foams separated into groups for comparison. Parts et al. [35] went one step further and instrumented the NBS smoke chamber for on-line continuous analyses for CO, CO_2, total hydrocarbon, oxides of nitrogen, oxygen, and hydrogen chloride. Hydrogen cyanide was measured at the end of some experiments.

O'Mara [36] carried out similar experiments in an instrumented smoke chamber, also selecting various toxic products for analysis. He studied the rate of formation of CO and CO_2 from various natural materials (mainly wood products). Analysis of various organic species were also carried out using gas chromatographic procedures.

The selected gas analysis approach has been used most extensively for fire toxicological hazard assessment. This approach assumes the hazard is due to a few selected toxic products and measures only these.

2.3.3. Selected Gas Analysis — Large Scale

A fairly sizable number of large scale fire tests have been carried out in the last few years only a few of which will be considered here [37-41]. The objective of most of these experiments was to determine the burning characteristics of materials under simulated-end use conditions and not necessarily toxic product hazard assessment. Certainly, toxicity hazard assessment was not the primary goal. However, selective gas (CO, CO_2, and O_2 and smoke measurements were made in most of the large scale experiments.

The Battelle [37] studies are most interesting since four different types of bedroom furnishings ranging from conventional to extensively fire resistive furnishings were used. These studies illustrate the effects of improved commercially available fire-resistive furnishings and "space-age" room furnishings over conventional materials in terms of fire spread and combustion product generation. Gas analyses show significant differences among the combustion products that were measured for the different types of furnishings. From the gas data the authors concluded that the products of major importance from a toxicological view appear to be HCN, CO, CO_2. The high aldehyde concentration in the "improved" room fire study, and the peak NO_x concentrations in all but the space-age room fire were recognized as significant hazards.

The goal of Southwest Research Institute [38] large scale fire tests was to determine the burning characteristics of upholstered furniture. Cigarettes were used as the ignition source. Combustion product measurements included CO, CO_2, and light obscuration at the 5-ft level. Oxygen depletion and temperature were measured in six locations.

Factory Mutual and Harvard [39] carried out bedroom fires to determine the sequence of events of such fires to guide other fire research efforts. Methenamine pills on a urethane mattress were used for an ignition source. Measurements of smoke (optically), oxygen, carbon dioxide, carbon monoxide and total hydrocarbon were made. Croce and Emmons [39] concluded that the gas concentrations in the lower half of the enclosure were such that escape was possible at any time prior to flashover.

Palmer et al. [40] carried out full-scale fire experiments to determine the burning characteristics of a variety of upholstered furniture and floor coverings arranged in a typical sitting/dining room. The upholstered furniture included traditional (wooden framing with mainly natural fibers), semi-modern (latex foam on wood), and modern (polyether based polyurethane foam on wood

and molded plastic) items. The conclusions that the authors reached are:

1. The peak concentrations of CO, CO_2, NO_x, and HCN were comparable for each furnished room.

2. The time required to reach maximum toxic gas concentrations decreased as furniture was changed from traditional to modern.

3. The rate of oxygen depletion was greater and the minimum concentration lower for modern furniture.

4. Of the 3 types of furniture, the rate of smoke production was least for polyether foam on wood, with the latex unit producing the most rapid smoke build-up.

5. Less time was available for escape in fires involving modern furniture than with the traditional furniture.

In the NBS floor covering studies [41], a 30-foot corridor 8 feet wide was used to assess the contribution of floor covering materials to the flame spread problem. Gas and smoke measurements were reported. Continuous measurements of CO, CO_2, O_2 depletion and smoke optical density were made. Samples of combustion products were analyzed. Extremely high levels of smoke and carbon monoxide were encountered in all the carpet fires. Extensive light attenuation occurred within five minutes after ignition. Carbon monoxide levels rose rapidly along with oxygen depletion in most cases so that it was difficult to determine which was the primary or most immediate hazard.

In view of the state-of-the-art, the large scale fire tests reported above are not very conclusive in terms of toxicological hazard. In a large scale fire the concentrations of the various toxicant species that one selects for analysis will be different depending on the sampling location. In addition the effects of multi-component toxicants on biological systems cannot be predicted at this time. The latter criticism can be applied to the extensive chemical analysis of small scale experiments.

2.4. Toxicity of Multi-Component Gas Mixtures

Statement of problem: What does one do with the large amount of analytical data?

Tsuchiya and Sumi [32] suggested that the toxicity of multi-component combustion products can be evaluated from quantitative data using the toxicity index (T) given by the relationship

$$T = \Sigma_i (C_e/C_f)_i \quad . \qquad (1)$$

In this relationship C_e is the concentration of a volatile or gaseous product evolved from one gram of material undergoing thermal degradation into a volume of 1 m^3. The term C_f is defined as the concentration of the gas that is fatal to man in 30 minutes.

The above relationship has been used for toxicological assessment. Woolley [31] calculated the toxicity index for the decomposition products from PVC. His chemical analytical efforts yielded approximately 75 products from the thermal degradation of a commercial polyvinylchloride sample.

Based on his chemical analysis, seven compounds were selected for calculating the toxicity index. These compounds were hydrogen chloride, benzene, toluene, total xylene, napthalene, vinyl chloride, and phosgene (used detection limit value). For these toxicants the C_f values were taken from the American Industrial Hygiene Association [42].[f] Estimated values were used when values were not available. Rashbash [43] suggested that one use twenty times the maximum allowable concentration (MAC) for the C_f values in the fire environment for a thirty-minute exposure.

Based on the above assumptions, Woolley [31] concluded that CO and HCl are the main toxic risks from decomposition products of PVC. However, information obtained by exposing animals to PVC decomposition products suggest more complex plenomena may be involved that cannot be explained by HCl and CO alone [76]. This will be discussed further in section 4.4.

It should be recognized that the above relationship is limited to the gaseous toxicants. The effects of smoke (particulates and aerosols) may modify any conclusions based on this index. In addition, the combustion gas mixture is so complex that minor products that are extremely toxic may remain unidentified and be the primary toxicological hazard or contribute to the toxicity in a significant way.

2.5. Limitations of Smoke and Toxic Gas Measurements

Statement of problem: Can one separate the smoke and gas measurements and determine the hazard?

As a result of the separation of the analysis of combustion products into two fractions (solid and gas) the inhalation or toxicological hazard has been separated in the same fashion leading to an over-simplification of the problem. Obviously the presence of smoke will reduce visibility (optical attenuation), but the irritant effects of the smoke resulting in tearing and burning of the eyes is most likely the primary limitation in escaping from a fire rather than light attenuation. An optical attenuation measurement cannot assess this problem. In addition, the adsorption of toxic species on the smoke particulates may be a primary mode of entry of toxic species into the respiratory tract. Desorption from the inhaled particulates is required, and the degree of toxicity from the desorption process is critical to the degree of toxicity. Obviously, an optical measurement and a strict chemical analysis of gases cannot assess this problem and one should be aware of this in assessing the toxicity hazard of combustion products.

A further word of caution is in order regarding the literature on the analytical assessment of the toxicity hazard. A number of the efforts to assess the toxicological hazard due to fire gases assumed that the hazard is due to CO, CO_2, O_2 depletion, HCl and HCN. Most of these gases are relatively easy to measure, except perhaps HCN. Based on this assumption, the approach to the toxicity hazard assessment has been to analyze only for these products and base the potential hazard on such measurements. This type of measurement neglects the possibility that other compounds may play a role in toxicity of environment. A hazard assessment derived from measurements founded on a questionable assumption is unreliable.

Some workers in the combustion field have not only assumed that these are the important gases but have gone one step further to assess the synergistic effect of a synthetic combination of these products [44]. This is being done even before effects of individual components are known to sufficient degree to allow recognition of the effects of multiple components.

It is interesting to note that Boettner et al. [33] concluded, after the analysis of the products from phenol formaldehyde, that no conclusions as to toxicity can be drawn pending further analytical work. After the analysis of the products from polyurethanes they conclude that carbon monoxide and hydrogen cyanide are the only acutely toxic compounds identified. Since carbon monoxide disrupts the ability of the blood to carry oxygen and cyanide disrupts the cell's ability to utilize oxygen, there is a toxicological synergism between the two. Is this a valid method of toxicity assessment and determination of synergism? What about the effects of nitriles and TDI identified by Woolley and Wadley [27] and Hileman et al. [28]?

Each of the materials that have been studied has an impressive list of products which depend on temperature and the environment of thermal decomposition. The relevance of these data to the toxicity hazard remains to be seen.

3. BIOLOGICAL ASSESSMENT OF TOXICITY

Statement of problem: How does one determine the effects of a complex mixture of toxicants on a complex biological system?

3.1. Background

Inhalation toxicity of combustion products is a complex problem involving a biological system, a combustion process, and analytical chemistry. This suggests that the problems associated with an investigation of the inhalation toxicity of combustion products can be divided into three major areas of expertise. A concerted effort to solve the problem must involve all three:

1. the methodology for the generation of the products;
2. the methodology for determining the biological response elicited from the exposure to the products;
3. chemical analyses (identification and quantification) of the products responsible for that response.

Some of the problems associated with determining the toxicology of combustion products have been discussed by MacFarland [45]. He compares conventional toxicology experiments to experiments on the inhalation toxicology of combustion products. In conventional inhalation toxicology, an organism is exposed to a known quantity of a single agent and the organism's responses are noted as a function of time and concentration (dose-response relationship). In combustion product inhalation toxicology, by contrast, the agents and dosage usually remain unknown due to the difficulty of measuring the amount of toxicants produced and inhaled, due to the large number of products produced, and due to the fact that the composition and concentration of products change with time and combustion conditions. In addition, as pointed out by MacFarland [45] and Amdur [46], particulates are also present and may play a significant role in the toxicity of the products that is difficult to assess analytically.

The toxicological studies on the thermal decomposition products of polytetrafluoroethylene (PTFE) as reported by Clayton et al. [11] and Waritz and Kwon [12] represent some of the most definitive work on toxicological assessment of thermal decomposition products of a polymeric material in which animals were used. These studies on PTFE are classic and illustrate the value of using animals as detectors for assessment along with some chemical analyses of products for correlation with the biological results. The chemical analysis was also used to verify the conclusion of the bioassay experiments. The importance of particulates in inhalation toxicity is also dramatically illustrated.

Zapp et al. [47] reported the pyrolysis products from PTFE decomposed at 300 °C in air to be tetrafluoroethylene, hexafluoroethane, hexfluoropropylene, octafluorocyclobutane, and octafluoroisobutylene. Clayton et al. [11] showed that a particulate material was responsible for the toxicity of PTFE at certain temperatures. Further work by Waritz and Kwon [12] showed that at a sample pyrolysis temperature of 450 °C, a 100% mortality was obtained on rats. By passing the effluent through microporous filters to remove particulates before exposing the animals to the products, the mortality was reduced to zero. This result suggested that the major toxicants reside in or with the particulate phase: at least at 450 °C. It was also noted that hydrolyzable fluoride was decreased by filtration. In addition, it was determined that the toxicity was strongly dependent on temperature and the atmosphere in which the material was decomposed, the toxicity being significantly reduced when decomposition occurred in a nitrogen atmosphere. When the rats were exposed to the degradation products generated at 450 °C in air, gross pathology showed microscopic focal hemorrhagic lungs. When pyrolysis was done in nitrogen no hemorrhages were observed in animals sacrificed after exposure.

Scheel, McMillan and Phipps [48] exposed rats to the pyrolysis products of PTFE containing hydrolyzable fluoride equivalent to 50 ppm COF_2 for one hour daily. Urine excretion of fluoride correlated with the measured changes in the succinic dehydrogenase activity as expected from fluoride poisoning. The authors concluded that COF_2 is generated during the pyrolysis of PTFE and hydrolyzed in the body fluids to produce fluoride toxicity in the exposed rats.

Scheel, Lane, and Coleman [49] exposed dogs, rabbits, guinea pigs, cats and mice to the pyrolysis products of PTFE. They also concluded that the principal toxic effects are due to the hydrolyzable fluoride produced when carbonyl fluoride and other hydrolyzable products are absorbed into the tissue. The lesions associated with the particulate material observed in the lungs of exposed animals prove that the particulate is irritating for at least seven to ten days following inhalation.

Various air pollution studies have also demonstrated the role of particulates in the inhalation toxicology of various materials and deserve some attention in fire gas research.

The role of an inert aerosol on the irritant properties of sulfur dioxide and formaldehyde has been studied in some detail by Amdur [46] using guinea pigs and deserves some attention in the fire gas problem. She found that 0.04 μ NaCl aerosol had a definite potentiating effect on the irritant properties of SO_2, where 2.5 μ aerosol did not. The effect increased as the aerosol concentration increased from 4 to 30 μg/ml at the same concentration of formaldehyde. The potentiating effect of the inert aerosol on the formaldehyde exposure was also particulate size dependent. This is consistent with the fact that the small aerosol with the absorbed gas will pass through the upper respiratory tract and reach the alveoli of the lungs where it is retained as an irritant and released causing deeper involvement of the lower respiratory tract. She also noted that the presence of the aerosol with irritant gas delays the recovery of the animal as compared to the exposure to the irritant gas without the aerosol. In the non-aerosol exposure, pulmonary resistance returns to pre-exposure value (level) in about two hours (depending on the concentration) whereas the recovery from the aerosol-irritant required a considerably longer time up to 24 hours. Amdur speculates that two physiological phenomena occur: 1) the low level irritant produces mechanical constriction which is reversible once the irritant is removed and 2) the combination of aerosol-irritant may be the beginning of tissue damage in the alveolar region of the lungs.

From the foregoing discussion it should be clear that particulates may affect the toxicological effects of combustion products on a biological system. It should be realized that the rate of desorption of any toxic

product adsorbed on particulates is critical. In turn, a number of factors will affect the products that are produced during thermal degradation. These include shape and weight of material, temperature of degradation, combustion equipment, air supply, and exposure chamber. In addition, the mode of degradation, flaming, smoldering, or pyrolysis will affect the composition and concentration of the products that are generated. Since the methodology for exposing the animals and assessing the effects on the animals will affect the conclusions, it is extremely difficult to compare the results found by various experimentalists who use different techniques. However, from a practical point of view, it may only be necessary to show that one material is more or less toxic than another under certain specified conditions.

3.2. System for Biological Assessment of Toxicity

Statement of problem: How does one generate combustion products reproducibly and expose animals to these products?

The last two years have produced a rapid increase in the number of laboratories that are doing biological testing of combustion products and the field is in a rapid state of flux. Table 1 is a summary of these systems presently being used for such purpose. Much of this work was reviewed at the Symposium on Toxicity and Physiology of Combustion Products [50]. Rather than reviewing and critiquing all of these experiments in detail, there are some general comments that are applicable to most of the systems that indicate the advantages and disadvantages of the various methodologies.

3.2.1. Combustion Systems

The nature and concentration of combustion products will depend on the (1) mass of the sample; (2) volume of the chamber; (3) heat flux or temperature to which the sample is exposed; (4) time of heat exposure; (5) sample formulation; (6) flaming or nonflaming mode; (7) atmosphere surrounding the sample; and (8) orientation and physical structure of the sample. Specification of these parameters is a minimum requirement if data are to be reproduced in the laboratory. This is not to suggest that other parameters will not become important as animal experiments are coupled with combustion experiments.

Various thermal decomposition techniques have been used to generate products for animal exposure in order to assess the toxicity of the gaseous products. The thermal decomposition systems fall into two broad classes: (1) those that generate the products external to the animal chamber with the products transferred to the animals (flow-through system); and (2) those that generate the products inside the animal chamber. Both have advantages and disadvantages that may affect the overall toxicological results.

An example of the first type of system is that being used in West Germany by Kimmerle [51] and Hofmann and Oettel [52]. This system has produced the most information since a wide variety of materials have been intercompared and evaluated on an equal volume basis and on an equal mass basis. According to Kimmerle [51], the German toxicologists have agreed on the methodology for the generation of pyrolysis products for the exposure. The thermal degradation system consists of a short tube furnace with the samples enclosed in a quartz tube. The furnace travels along the quartz tube — sample, with air flowing in the direction of furnace travel or counter to it. Using this system the toxicity of the products can be compared by sample weight or sample volume. Kimmerle states that "for an evaluation of combustion processes, the second approach (volume) seems to be more in line with actual conditions" and, furthermore, that in Germany "it was agreed that only

smoldering conditions are of interest." It would appear to this author that the decision on smoldering combustion is a bit premature until more information on the relative toxicity of the products of smoldering combustion as compared to flaming combustion is available as determined by careful bioassay experiments.

The efforts in France [53] and Belgium [54] summarized in Table 1 use the German system for combustion product generation. The work in France was reported in some detail at the International Symposium on Physiological and Toxicological Aspects of Combustion Products in March 1976 [53].

The second type of system is used at the University of Utah in which animals have been placed directly in a smoke density chamber [14]. This system specifies many of the parameters that influence the quality and quantity of products. Moreover, with recent modifications reported by Seader, Chien, and Birky [55], it is possible to change heat flux from 2 to 7.5 W/cm^2 and monitor sample mass on a continuous basis. The chamber, however, is not the most convenient for animal exposures in terms of observations of the animals and monitoring animal physiological functions. These limitations will be discussed later.

3.2.2. Animal Exposure System

Even though it may not be possible to determine a true dose in fire experiments, the time of exposure must be specified. Whether or not the animals are restrained or allowed to move about during the exposure may have a significant effect on the outcome of the experiment. Animals that are free to move about in a cage generally will huddle together causing variations in exposure.

Placing the animals directly in the chamber where combustion occurs or transferring the combustion products to the animal chamber may also cause significant differences in the results. If the products are transferred into the animal chamber then the temperature, diameter and length of the transfer lines can play a role in the dosage received by the animals. In this case, it may be very difficult to intercompare biological results with other experiments and to intercompare the toxicity of products from different materials due to the preferential trapping of different constituents. If the aerosol or particulates play a significant role in the toxicity of PVC then the assessment of the toxicity of PVC relative to the toxicity of some other product that does not involve particulates will be misleading. For example, the studies on the fire retarded rigid polyurethane foam [14,15] mentioned earlier indicate that the highly toxic bicyclic phosphate ester may be concentrated in the smoke (particulate) phase of the pyrolysis products.

In the flow-through or product transfer system, a toxic product that has a relatively low vapor pressure may be trapped out on the walls of the transfer tube unless the walls are heated. This differential trapping can make a significant difference in the ranking of materials for toxicity hazard. If the material produces low levels of a highly toxic agent it may not appear to be hazardous when the products are transferred through a cold tube.

In the systems that place animals directly in a combustion atmosphere, such as the smoke density chamber, one must be aware of the potential for producing a decreased oxygen atmosphere and elevated temperatures. This, of course, depends on the ratio of chamber volume to the sample mass. Both oxygen concentration and air temperature should be measured in order to avoid placing these stresses on the animal. In addition, animal monitoring for EEG and EKG is difficult in the smoke density chamber as presently designed since it was not made for this purpose. As a result of this new application of the smoke density chamber, modifications for animal exposures are being developed.

In the German system, large amounts of air are mixed with the combustion products so that decreased oxygen content is not likely to occur and elevated temperatures are not likely to be a problem. Both of these stresses (high temperature and low oxygen) will cause major differences in toxicological results.

Cornish [56] in a corollary study to that of Boettner et al. [33] ranked materials according to material LD_{50} (lethal dose of thermal degradation products resulting in 50% mortality) using two different systems for product generation and exposing the rats. He referred to the two systems as a "flow-through chamber" and a "static chamber." He concluded that the relative toxicity of the thermal decomposition products of a number of materials is significantly different as determined by the two systems. This is not a surprising result when one reviews the two systems.

In his flow-through system, a pyrolysis mode of thermal decomposition was used; and in the static chamber, a combustion mode was used. It is not entirely clear if combustion occurred in the flow-through technique; but at a heating rate of 3 degrees per minute, it is unlikely. In the static system, the sample combustion temperature was reached in one or two minutes.

In addition to this rather fundamental difference, exposure time and product concentrations were also different. Pyrolysis in the flow-through system was continued for 140 minutes with an airflow over the sample of 1 liter per minute diluted with 2 liters of air per minute before exposure. This gives a total volume of approximately 420 liters and an animal exposure time of 140 minutes. In the static chamber the total volume was 1500 liters and the exposure time was four hours.

Both exposure times are unrealistic for exposure to combustion products. For the fire environment, a maximum exposure time of 30 minutes is reasonable. In addition, if the rate of intoxication is an important parameter, then, of course, the exposure time and sample mass to air volume ratio (fuel loading) and heating rate and combustion rate become important parameters.

If the goal is to compare the two types of animal exposure techniques then the products should be generated in the same fashion. If the goal is to compare pyrolysis products with combustion products, then of course the same animal exposure conditions must be used. But when both the thermodegradation conditions and animal exposure conditions have been changed simultaneously, one cannot determine what effect is related to which variable. As a result of measurement with two entirely different systems, the fact that Cornish [56] ranked various materials in a different order based on a lethal dose per gram of material undergoing thermal decomposition means only that the results depend on the system as pointed out previously.

3.2.3. Chemical Analysis Systems

The analytical measurements can be divided in two categories: (1) analysis of combustion products (environmental) to which the animals are exposed and (2) biochemical analysis, predominately blood chemistry. Measurements and correlation of toxicants in environment and in the biosystem have not been done to any great extent in the biological assessments. This approach was used in studies on polytetrafluoroethylene [47-49].

The various degrees of chemical analysis of combustion products were discussed in earlier sections. Measurements of selected products discussed in these sections are extremely valuable in animal experiments. Oxygen measurements are necessary to determining if oxygen depletion is a significant factor or in order to avoid this condition. Carbon monoxide, a toxicant produced in most if not all fires, should be measured and correlated with the COHb levels in the animals. Carbon dioxide is also common to the combustion

process and should be monitored. If hydrogen cyanide is considered to be a product in the degradation of a material, then one may wish to monitor this toxicant.

In some of the systems that have been reviewed in the previous sections [51,53,54], carboxyhemoglobin data is reported either at the point of death, incapacitation or at the end of the exposure period. This data is extremely valuable in determining the significance of carbon monoxide relative to some other toxicants. That is, if the value of COHb is significantly less than that required to cause incapacitation or death and the animals were either incapacitated or dead, then some other toxicant should be considered. This approach was used in the study reported by Petajan, et al. [14].

The chemical make up of the polymer system being studied can be used to determine what other toxicants should be measured in the biological system. For example, blood cyanide can be measured when the animals are exposed to degradation products from polymers containing nitrogen in their chemical structure. This measurement may cause some difficulties which are discussed below (section 4.2). In any event, COHb measurements appear to be minimal biochemical measurements.

4. MECHANISMS AND MODELS

Statement of problem: Very little is known about the mechanism of action of most toxicants found in fire environment except CO and perhaps HCN. Most toxicity studies have been directed to chronic (long term, low level) exposures to single toxicants. Virtually nothing is known about the effects of complex mixtures of toxicants found in the fire environment or about the effects of short exposures to high concentrations or single toxicants.

Various reviews on toxic products in the fire environment have included reviews that describe the expected physiological effects of individual toxicants at various levels on humans [51,57]. Some animal reponse data is also included in Kimmerle's review [51]. All of the information presented in these reviews is limited to a description of physiological symptoms and does not consider mechanisms. The effects of multiple components have not been investigated and it is clear that multiple component effects cannot be determined without a clear understanding of the effects of individual toxicants.

4.1. Carbon Monoxide

The physiological effects of low levels of carbon monoxide on man and animals have been studied extensively [58,59]. The mechanism of intoxication is well documented and is primarily the result of tissue hypoxia caused by the inability of the blood to carry sufficient oxygen to the cells [60]. The inability of the blood to transport oxygen is due to the fact that the affinity of hemoglobin for carbon monoxide is 300 times greater than that for oxygen so that the CO preferentially ties up the hemoglobin to produce carboxyhemoglobin (COHb).

R. Stewart et al. [61] exposed humans to high concentrations of CO for short periods of time and monitored EEG, EKG, cardiac output, COHb, visual evoked response, etc. The main objective of this study was to accurately determine the rate of CO absorption during brief exposures to the gas in concentrations found in automobile exhaust and in burning buildings. Since COHb levels were not allowed to exceed 16%, significant changes in the measured physiological parameters did not occur.

As a result of these and other studies on carbon monoxide it is well known at relative low concentrations what level of carbon monoxide in the environment will produce a given level of COHb and furthermore what the physiological response is likely to be. It is not as well known what happens on exposure to high concentrations of CO that result in corresponding high carboxyhemoglobin concentrations in human subjects. Likewise, it is not known if individuals with cardiovascular disease are more susceptible to the effects of carbon monoxide. In addition it is not known what the effects of CO are on an individual who has a high level of blood alcohol.

Based on the information on the toxicity of carbon monoxide, the toxicity of combustion products from wood was compared to that of CO by Edginton and Lynch [62], and Zikria [63]. Edginton and Lynch concluded that the sole toxic principle released in hazardous amounts by burning plywood or flame retardant treated plywood is carbon monoxide.

Zikria noted that when test animals were exposed to the combustion products from wood, carbon monoxide alone did not account for the deaths. By combining gas analysis with bioassay experiments and with clinical data that showed many deaths involved respiratory tract damage, he concluded that aldehydes in combination with the CO could explain the deaths.

Carbon monoxide has become the toxicant of choice for use in the development of an animal model for toxicity assessment of combustion products [64]. This is due to various reasons. First, CO is believed to be produced in virtually all fires. Secondly, it is probably the most studied and understood toxicant and consequently its effects on many species is well documented making it relatively easy to compare CO toxic response to the response from combustion products.

4.2. Hydrogen Cyanide

Statement of problem: What is known about role of HCN in fire deaths?

Hydrogen cyanide is a good example of a toxicant about which there is a lot of speculation in fire literature and not much factual information. It has become a major concern in fire fatalities since (1) it is known to be a product resulting from the thermodegradation of some polymers containing nitrogen, (2) because it has a relatively high toxicity, and (3) there is a strong public reaction to this toxicant. The role of hydrogen cyanide in fire fatality studies raises some interesting questions since most of the data on cyanide in these studies are questionable due to questionable sample treatment and analytical procedures. At this time there is little clinical information or data from animal studies to determine the validity of this concern. The data that are available raise more questions than they answer.

For a detailed discussion of the mechanism of action of cyanide on biological systems the reader is referred to Goodman and Gilman [65]. Hydrogen cyanide reacts readily with trivalent iron of cytochrome oxidase in mitochondria to form the cytochrome oxidase-CN complex which inhibits cellular respiration, an action referred to as cytotoxic hypoxia. In low concentrations, cyanide stimulates respiration. Its activity on the brain electrical activity

is one of depression. Lethal concentrations result in progressive slowing of the heart and the heart continues to beat several minutes after the last respiration.

The physiological effects of cyanide on the central nervous system and the cardiovascular system have also been delineated. The route of detoxification is through the formation of thiocyanate by the action of rhodanese. Detoxification is limited by the endogenous supply of thiosulfate.

What does not appear to be well known is the level of environmental cyanide that will produce a given biological or blood level, and what will be the corresponding physiological effects in humans or animals at a given level. A great deal of the difficulty appears to be due to the lack of accurate and reproducible analytical techniques for the measurement of hydrogen cyanide in air and in biological systems.

Wetherell [66] determined blood cyanide levels in 53 fire victims in Michigan and found that 39 of the victims had blood cyanide levels that ranged from 17 μg/100 ml to 200 μg/100 ml. The minimum lethal level is reported to be 300 μg/100 ml [67]. All measurements were performed within 24 hours on refrigerated samples so that cyanide from decomposition was supposedly ruled out. Also stomach cyanide levels were determined to rule out ingestion.

Post mortem analysis of 26 victims of cyanide poisoning by Sunshine and Finkel [68] shows that the brain has the lowest cyanide levels and the spleen the highest. They found that in some refrigerated samples, blood cyanide concentrations rose to 500 to 700 μg/100 ml in 14 days. They also demonstrated that blood samples incubated at 37 °C did not show cyanide. The source of the cyanide in the refrigerated samples was believed to be from bacteria. This information raises some questions about the significance of Wetherell's data and conclusions.

The problems of sample handling and other questions concerning the analysis of HCN in biological samples need to be answered before significance can be attached to clinical cyanide measurements. Not only is the treatment of biological samples for cyanide analysis important, but the time elapsed between death and sample collection must also be considered. Obviously even before these questions can be answered, a good reliable analytical procedure must be available for cyanide analysis of biological samples.

In summary, the role of hydrogen cyanide in fire fatalities or casualties is not clear. It is known that some hydrogen cyanide is produced when nitrogen containing polymeric materials are exposed to or involved in fires, but until more is known about its significance in human casualties, its hazard in fires cannot be stated.

4.3. Synergism

Statement of problem: What do we know about the synergistic effect of two or more toxic gases in fire deaths?

The possibility of synergistic action between two or more toxicants in the fire environment has been suggested by a number of individuals in the fire literature. Synergism in the toxicological sense is defined as a toxicological response from a mixture that is greater than the summation of the individual toxicities of the components. For example Boettner et al. [33]

concluded that carbon monoxide and hydrogen cyanide are the only acutely toxic compounds identified from the analytical studies on polyurethane; and since carbon monoxide disrupts the ability of the blood to carry oxygen and cyanide disrupts the cell's ability to utilize oxygen, there is a toxicological synergism between the two.

Moss et al. [69] exposed rats to CO and HCN alone and in combination to determine if synergism exists between the two toxicants. Although they avoided the term synergism, their descriptive conclusions are suggestive of the fact that they considered the toxicological interaction between CO and HCN to be more than additive.

Based on exposure of rats to CO and HCN, Kimmerle [51] concluded that there was a synergistic action between the two toxicants. Birky [70] analyzed the data obtained by Kimmerle and concluded that synergism was not demonstrated.

In an effort to determine if toxicological synergism does occur between CO and HCN, Lynch [71] exposed rats to CO and HCN singly and in combination. Death was used as the endpoint. Based on this work, Lynch reported that synergism did not occur between these two gases.

Pryor and coworkers [72] investigated the combined actions of various factors related to the fire environment. They exposed mice to the combined actions of oxygen depletion, carbon dioxide, carbon monoxide, and heat as the primary variables. Low levels of hydrogen cyanide, nitrogen dioxide and sulfur dioxide were added singly to the above constituents to study the combined action.

It is difficult to assess the data presented by Pryor and coworkers on the effects of these combinations of noxious gases due to the large variability of the data. For example, in their table 4, three mice died as a result of a four-hour exposure to 16% O_2, 0.075% CO, and 30% CO_2 at 85 °F, while in their table 7, no mice died when exposed to the same atmosphere at a temperature of 100 °F for 4-hour and 24-hour exposure times. The authors suggest at one point that a new batch of animals gave different results. However, it is not clear why this should be so, or whether the data in table 7 are comparable with the data in table 4. In any event, the large variability shown by these data suggests that the conclusion regarding synergism due to low levels of irritant gases is highly questionable. In addition, an exposure time of four hours is excessive for relevance to the fire problem.

It is not entirely clear that the final answer is available concerning the question of synergism between CO and HCN. Data that are available suggest that synergism does not occur between these 2 toxicants, at least in rats. In one sense synergism has become a catchword to explain many unanswered questions regarding fire fatalities.

4.3.1. Mathematical Analysis for Synergism

A mathematical equation was defined in section 2.4.3 as a means of estimating the additive effects of a mixture of toxicants. It has been proposed [70] that this equation can be used to determine if two or more toxicants are additive or synergistic in nature. In equation (1), if C_f is defined as the concentration that is fatal to 50% of the animals (LC_{50}) for the individual toxic components and C_e is the concentration of the individual components to which the animal is exposed in a mixture, then the sum of toxicity indices (T) should be approximately 1 at the LC_{50} concentration of the combination if no synergism exists; that is, the toxicity of two or more agents is additive.

$$T = \Sigma_i \ (C_e/C_f)_i = 1$$

If the summation is less than 1, then it can be assumed that synergism exists and in the summation above an interaction term is required. On the other hand, if T is significantly greater than 1, then one should look for antagonism.

Kimmerle [51] has some data on the toxicity of carbon monoxide and hydrogen cyanide that served to illustrate the use of the above relationship.

According to Kimmerle, the 30-minute LC_{50} for rats exposed to carbon monoxide and hydrogen cyanide individually are 5500 ppm and 200 ppm, respectively. In table 2 reproduced from Kimmerle [51], 3300 ppm of 30 and 45 ppm of HCN resulted in 50% mortality again for 30 minutes. The toxicity index for this combination is

$$T = 3300/5500 + 45/200 = 0.83$$

Similarly, 4400 ppm of CO and 38 ppm of HCN gave a 45% mortality, not quite 50% but probably within experimental error. These concentrations give a toxicity index of 0.99.

By the above stated criteria, one might expect that some synergism exists in the first case since $T < 1$ (0.83). However, the uncertainty in the overall measurement, while not stated, is expected to be in the order of ± 0.2. This would include the errors in HCN and CO measurements and variability in animal response. The conclusion of this author is that no synergism has been proven to exist between HCN and CO, at least by these data, contrary to Kimmerle's conclusion. Certainly the second set of data where $T = 0.99$ shows no synergism by the above criterion.

4.4. Sensory and Respiratory Irritants

Statement of Problem: What is the relative role of eye and nasal irritation as compared to light obscuration during fire?

As indicated in earlier sections, light attenuation measurements have been used exclusively as a measure of the hazard of smoke from combustion products. While it is true that light attenuation is a problem for the firefighter both in fire fighting and rescue operations, inability to find an exit due to impairment of vision caused by irritants may be the primary problem for the individual caught in a fire.

The effects of irritants in smoke on visual acuity was investigated by Lopez [73]. He studies smoke emission from burning aircraft cabin materials to determine: (a) the degree of correlation between NBS smoke chamber data for various cabin materials and similarly acquired data from large-scale tests conducted in a cabin mock-up representative of a wide-bodied transport, and (b) the effect of smoke on human visual acuity and the relationship of this visual acuity to optical attenuation measurements. For the second objective, two sets of human subjects were used: (1) individuals outside the mock-up viewing a sign through a window and (2) the second set in which the window was removed so that their eyes alone were exposed to the smoke.

Ten classes of cabin materials were tested with two different compositions of each class. For example, a wool carpet and a 50/50 cotton-rayon carpet were tested. Likewise two Tedlar coated ceiling panels were tested.

The major conclusions of the study were that: (a) fair to good correlation exists between laboratory chamber and mock-up smoke measurement results, and (b) that the dominating factor on human visibility is the irritating effects of combustion products as opposed to light obscuration.

An extensive review of the mechanisms of sensory irritation in the upper respiratory tract was prepared by Alarie [74]. The nasal respiratory epithelium, as well as the cornea, receive their sensory innervation from the afferent trigeminal nerve. As pointed out in this review there are some important differences between the sensory nerve endings of the nasal respiratory mucosa, the cornea, the skin and other mucous membranes. "However, all represent the common chemical sense receptor in mammals and in general can be stimulated by the same chemicals."

Based on this knowledge of the basic physiological similarities between the sensory nerves, Alarie reported on the irritant properties of combustion products from a polyurethane using mice as experimental animals. Change in respiratory rate and tidal volume was measured and expected human responses proposed [75]. Table 3 taken from Alarie's results, is the proposed relationship between results obtained with mice and the proposed human response to sensory irritants. In a comparison of the sensory irritation response in mice to HCl and Cl_2 and the thermal degradation products of PVC, Alarie concluded that the decomposition products of PVC were somewhat more potent than HCl [76]. No explanation of this fact has been obtained at this time, but this fact would tend to be born out by experience of the fire services in fighting electrical fires. Fires involving PVC electrical insulation, as reported by Dyer and Esch [77], appear to be deceptively hazardous.

4.5. Organophosphate Ester Toxicity

Statement of problem: Is the identification of the highly toxic phosphate ester in the combustion products of a fire retarded polyurethane foam predictive of future problems with fire retarded materials?

Since many of the fire retardants used in polymeric materials are organophosphates or halogenated organophosphates, this raised the obvious question of the toxicity of these compounds, both in the environment and of their combustion products during a fire.

4.5.1. Neurotoxicity of Organophosphates

Two extensive reviews on the mechanism of neurotoxicity of chemical agents and in particular on organophosphorus esters have been prepared in the last couple of years [78,79]. This most recent review is oriented to the biochemical mechanism of neurotoxicants [79]. The subject is extremely complex and in some cases the mechanisms are not clearly understood. For an understanding of what is known about the mechanism of action, the serious reader is referred to the above references.

In summary, the toxic actions of organophosphorus compounds are of three known types: (a) acetycholinesterace inhibitor, (b) delayed neurotoxic action and (c) γ-aminobutyrate antagonism (GABA). The first two types are well documented in terms of clinical manifestations, but it is only the first type in which the biochemical mechanism is well understood. In those compounds that show delayed neurotoxic activity, the action is postulated to be by inhibition of esterases. The esterase activity is referred to as "neurotoxic esterase" for which the physiological function and biochemical mechanism are unknown. The third type of action, GABA antagonism, is not discussed in the above reviews and very little exists in the literature on this type of toxic action.

The review of the neurotoxicity of organophosphorus esters cover the first two types of neuropathies [79]. The first is a selective effect upon certain hydrolytic enzymes, notably pseudo (or serum) cholinesterase. This type of action is most commonly referred to as acetycholinesterase inhibition. As mentioned, the biochemical mechanism is well understood and the clinical symptoms are quite characteristic.

The second type produces a delayed neurotoxic sequelae in which the clinical effects are not seen until 8 to 14 days after ingestion of the toxic agent. The clinical manifestations of the intoxication are well known and these will be described. However, as indicated, the biochemical mechanism is not well understood.

A comparison of a number of organophosphates shows, however, that there is no correlation between the neurotoxicity with inhibitory power against the esterases, an enzyme in the brain and spinal cord that is the target for neurotoxic agents. The biochemical mechanism is believed to be by inhibition of neurotoxic esterase even though it is known that young hens do not develop neuropathy after single doses of neurotoxic agents; though their neurotoxic esterase is inhibited. This is apparently an unresolved problem and possible suggestions for the lack of neurotoxic response have been made in young chickens. In general, the young in most species seem to be more resistant than the adults to the effects of delayed neurotoxicants.

By way of physiological mechanism of the delayed neurotoxicants, the following observations have been made. By far the largest number of chemicals with selective neurotoxic activity appear to act upon the neurons. The neuron has two properties not found with other tissues that render it more sensitive to outside influences. First, neurons that are irrepairably damaged and die cannot be replaced in mature animals. Any "recovery" from loss of neurons can only be effected by establishing new neuronal circuits or by greater use of those remaining. Secondly, if the structural maintenance program of a neuron is interfered with so that it is unable to maintain its long axon, and consequently degeneration of a distal part of the axon occurs, a break in communication system takes place leading to a loss of function of the signaling unit. By comparison, if a break occurs in the peripheral nerve fibers regardless of the extent, the potential for regeneration is very great and contact is re-established although the growth rate is limited to about 1 mm/day. Repair of damaged central nervous system fiber does not occur.

It is observed that while the initial attack on neurotoxic esterase occurs throughout the nervous system, only certain axons degenerate. This is believed to be related to the length and diameter of the axon, the longer axon being affected to a higher degree.

One can summarize by saying that "at present no clear metabolic disturbance has been proven to be associated with the inhibition of neurotoxic esterase and the genesis of the delayed neurotoxic response." As a warning, delayed neurotoxicity is an irreversible effect, and the fullest possible measures should be made of the hazard associated with a new compound.

There is another class of compounds that compete for the active site of the neurotoxic esterase that are not only non-neurotoxic but actively protect test animals from the neurotoxic phosphate esters. Carbamate, phosphinate and sulphonate produce inhibited enzyme in which there is no toxic effect and protection occurs since the enzyme is not available for attack by neurotoxic compounds. These compounds can be used as blocking agents.

In the delayed neuropathy, the onset of paralysis in man is signaled by abnormal sensory experience such as burning or tingling, particularly in the feet and hands. Motor weakness is the cardinal symptom that begins in the feet and later involves the legs more generally and the hands. Ataxia may develop depending on the degree on intoxication. Sensory nerves may also

show degeneration, again particularly in distal limb regions; the most widely affected end-organ being the annulospiral formation of the muscle spindle.

Man and the chicken appear to be equally susceptible to the neurotoxicity of organophosphate esters and the latter has become the standard test animal. As noted earlier, the young of a sensitive species are usually much less susceptible and the reason for this is not known. Consequently, adult hens more than 100 days old are used for screening of O-P compounds. Rats and guinea pigs seem to be insusceptible or respond only poorly.

Tri-o-cresyl phosphate (TOCP) is the classical example of a phosphate ester that produces a delayed neurotoxic response, and it has been studied in some detail. Cresyl and related phosphates have been used as hydraulic fluids, lubricants, plasticizers and flame retardants. Studies on these types of compounds show that a large number of triaryl phosphates possess neurotoxic potential, although only a few limited correlations could be made between structure and activity.

Biochemical studies on TOCP show that a liver metabolite of TOCP is 30 to 60 times more neurotoxic than TOCP itself. Based on this finding it was suggested that, as far as aryl phosphates are concerned, the liver plays an essential role in producing the toxic substance. It is believed that this process helps to explain the longer delay period between the administration of an aryl phosphate and the onset of symptoms of paralysis as compared to the alkyl phosphates.

Extensive studies on a wide range of alkyl and aryl-substituted analogues have shown that the only thing common to the neurotoxic and anti-esterase compounds is possession of a phosphate group. There is thus at present no certain way of predicting on the basis of its chemical structure whether a compound will be neurotoxic or not until tested.

It was reported that triphenyl phosphate was neurotoxic in cats, producing an effect like TOCP. It has since been shown that pure triphenyl phosphate is totally non-neurotoxic in the hen. Samples of "pure" triphenyl phosphate prepared from coal-tar phenol have been shown to contain neurotoxic impurities, as have other triaryl phosphates.

This type of information should serve as a warning to those laboratories doing research on existing and potential fire retardants that are organophosphates that may in themselves be non-neurotoxic but may contain impurities that can be very neurotoxic.

Two organophosphate compounds used for fire retarding textiles have been reported to possess anticholinesterase activity [80]. In this study anticholinesterase activity of tris (2,3 dibromopropyl) phosphate (I), N-methylol dimethyl phosphonopropionamide (Pyrovatex CP) (II), and THPOH [(Tetrakis (hydroxymethyl) phosphonium hydroxide] (III) were compared to the insecticide, Tetram S-[2-(diethylamino) ethyl] (0,0-dithyl phosphorodithioate hydrogen oxalate). The first compound (I) was found to produce about 16% of the inhibition of the insecticide whereas compound II produced 25% of the inhibition of the insecticide on a equimolar basis. The anticholinesterase activity of compound III could not be determined due to measurement interferences.

In the same study goldfish were exposed to 1 ppm in 30 gallons of water of each of these three compounds individually. All the fish died within five days from compound I, none died from the second fire retardant in a 30-day exposure, and 5 of the 6 fish died within 16 days from compound III.

Skin absorption of fire retardants as a result of wearing flame resistant treated fabric has been a concern for a number of years, although there appears to be little information available on this subject. Tris (2,3 dibromopropyl) phosphate (TBPP) has been studied to a limited degree [81]. Chromatographic analysis of treated urine from rats exposed to dermal applications of TBPP yielded a 2,3 dibromopropanol free and conjugated (DBP) that was believed to be a metabolite of TBPP. This compound was not found in the urine of individuals wearing clothing treated with the fire retardant although a rat allowed to chew on such a fabric did have low levels of DBP.

4.5.2. Carcinogensis of Organophosphate Fire Retardants

Tris (2,3 dibromopropyl) phosphate was tested [82] for mutagenicity in the histidine deficient strains of Salmonella typhimurium according to the Ames procedure [83]. Preliminary results of this testing indicate that this compound is mutagenic and that metabolic activation by rat liver enhanced the mutagenic activity. The Salmonella/microsome test shows that 90% of the known carcinogens were detected as mutagens. The reverse correlation has not been established. Carcinogenicity studies on rats and mice are in progress at the National Cancer Institute to validate the above findings.

The first indication of a potentially serious problem in the fire environment due to organophosphate compounds was reported by Petajan et al. [14]. It was shown that a highly toxic bicyclic phosphorus ester (4-ethyl-1-phospha-2,6,7-trioxabicyclo (2,2,2) octane-1-oxide) was produced when a phosphorus fire retarded rigid polyurethane foam based on a low molecular weight propoxylated trimethylolpropane was exposed to a high heat source and thermally degraded. It was reported that the rats exposed to the combustion products from this foam exhibited major motor seizures and died of status epiliptus.

The toxicity of the 4-alkyl bicyclic phosphorus esters was previously reported [16] at which time it was noted that the toxicity syndrome in mice and rats from an acute exposure do not resemble the characteristic poisoning by acetylcholinesterase inhibition. The mechanism was not defined.

These data suggested that a third type of neuropathy attributed to organophosphates that was not addressed in the review articles above may occur. The mode of action of the third type was defined by Bowery et al. [84] as γ-aminobutyrate antagonism.

The above limited data on the toxicity of organophosphorus compounds used as fire retardants should not be interpreted or extrapolated to the point of suggesting that all fire retardants enhance the toxicity of combustion products or of the materials in which they are included. In fact, quite to the contrary, preliminary results from a bioscreening program at the University of Utah in which thermal degradation products from polymeric materials with and without fire retardants are compared do not show any significant change in the toxicity of the combustion products from these materials. This is not to suggest that there will not be materials in which the toxicity of the fire retardant is unacceptable or that the combustion products of a polymer with a fire retardant will not be increased as compared to the material without the fire retardant.

5. PREVENTIVE MEASURES THROUGH CODES AND STANDARDS

Statement of problem: Can building codes and standards be used to limit smoke and toxic products?

5.1. Building Codes

Combustion toxicity requirements for cellular plastics appeared first in the 1958 Uniform Building Code. In the 1961 Code the toxicity requirement was extended to other interior finish materials. The requirement states: "The products of combustion shall be no more toxic that the burning of untreated wood under similar conditions." This requirement has remained unchanged at this time.

The toxicity code creates an interesting situation since at the time it was written the toxicity of wood was basically unknown and there existed no recognized test method for its determination or for the comparison of wood with a plastic material. This situation is still true. As a result the requirement has not been enforced or perhaps it is more appropriate to state the requirement is unenforceable.

As a result of this situation the International Conference of Building Officials research committee proposed an acceptance criteria for foamed plastics under Section 1717 of the 1976 Uniform Building Code that included a biological test method using rats by which wood combustion products are to be compared to the plastic material [85]. This toxicity requirement was dropped. This is the status of toxicity requirement in the building codes today.

Smoke density requirements were introduced in the 1961 edition and were also related to the burning of untreated wood under similar burning conditions. Methods are available however for smoke measurements based on light attenuation. The relevance of such a measurement to physiological hazard is however open to considerable question.

The French government recently passed a toxicity regulation limiting the use of synthetic materials containing chlorine and nitrogen in their polymeric structure [86]. The limitation applies to synthetic materials for use in public establishments that do not receive M_o and M_1 flammability ratings. The toxicity requirement applies only to the M_2, M_3, M_4 and M_5 classification as determined by the fire test "a'l' epiradieur" or radiant heat exposure. Materials included in the regulation are: decorative elements, wall hangings, door hanging, curtains, wall coverings, partitions, ceiling, ducts and pipes not built in, built in furniture, and furniture fixed in place.

Only two toxic gases, HCN and HCl, are considered in the regulation. The total quantity of nitrogen in the synthetic material is limited to 5 grams per m^3 of the volume of the buildings, while the chlorine in a synthetic material is limited to 25 grams per m^3 of the volume of the buildings.

Permissible quantities of the materials were determined by the following criteria or assumptions:

1. one third of the material will be exposed to degradation temperature prior to evacuation during fire,
2. one third of this fraction will give rise to an emission of nitrogen as HCN and chloride as HCl,
3. toxicity limits for fatality were set at 450 ppm for HCN and 1500 ppm for HCl, and
4. HCl concentration limit doubled due to its early detection as a result of the fact that its irritant properties will warn people of the hazard.

This legislation went into effect in March 1976. This appears to be the first attempt at integrating toxicity hazard with flammability criteria.

5.2. Code Requirements for Detection and Suppression System

The use of various fire protection systems in buildings can substantially reduce or eliminate the exposure of the building occupants to the toxic products of combustion. For this reason many codes require the installation of detection, fire suppression and smoke control systems in buildings. The National Fire Protection Association Life Safety Code [87] for example, requires the use of detection and extinguishing systems in such occupancies as newly constructed hospitals and nursing homes. The model building codes such as the BOCA Basic Building Code [88] require complete automatic sprinklers in high-rise buildings. Many state authorities such as Maryland and Massachussetts, currently require the installation of smoke detectors in all new one- and two-family dwelling construction.

5.2.1. Fire Detection Devices

From the moment of its initiation, fire produces a variety of changes in the surrounding environment. Any product of a fire which changes the ambient condition is referred to as a "fire signature" and has the potential for use in detection [89]. The production of smoke for example will result in a decrease in visibility. Not all fire signatures however, are practical for detection purposes. To be useful, a fire signature should generate a measurable change in some ambient condition. The magnitude of that change must be greater than the normal background variations for the condition. The fire signatures currently most practical for use in detecting fire in non-industrial occupancies are the aerosol signatures and the convective thermal energy signature. The aerosol material released during fire is generally referred to as smoke. Smoke appears in detectable amounts early in the history of a fire. Work on the siting of smoke detectors for residential applications by Illinois Institute of Technology Research Institute [90] has indicated that properly placed smoke detectors in a dwelling can provide the occupants with sufficient time to leave the building prior to the accumulation of untenable amounts of smoke or toxic gases.

The convective thermal energy signature is commonly detected by the use of heat sensors. Although heat is also generated in the early stages of the fire growth scenario, considerable time is often required before detectable heat levels are present, even in the room of fire origin. In several full-scale fire experiments it was shown that untenable conditions were reached in exit passageways prior to an alarm from heat detectors located in the room of fire origin [91]. The use of early warning detection devices to provide time for building occupants to escape the effects of smoke and toxic products offers one possible supplement to the control of toxic gases by materials standards. It must be remembered however, that detection systems simply provide a warning of potential hazard and it is therefore incumbent upon the occupants themselves to take some positive action to reduce their exposure to toxic products once the alarm has sounded.

5.3. Automatic Sprinkler Systems

Automatic sprinkler systems can reduce the occupant exposure to toxic products of a fire by eliminating or substantially reducing the fire at its source. The overall sprinkler performance record to the United States [93] indicated that they are 96.2% effective across the total range of occupancies. Similar information obtained in Australia and New Zealand [94] shows an overall performance record of 99.76% satisfactory. The reason for the discrepancy lies in the fact that in the United States, fire controlled by one or two sprinklers are seldom reported to the National Fire Protection Association. In Australia and New Zealand it is required that all sprinkler systems be connected to the fire department and therefore even single head

operations are reported in their data bank. It should be noted however that the typical sprinkler system is heat actuated and therefore will be subject to the same delays and reponse to a fire that are noted with typical heat detectors. Response can be speeded up considerably by using a smoke detector to initiate waterflow but this poses problems with false alarms [95]. Work is currently underway, however, to develop automatic sprinklers with a more rapid response to heat. A series of test methods and performance criteria is currently under development for such devices by Underwriters' Laboratories.

5.4. Smoke Control Systems

Under some circumstances, in high-rise buildings for example, the use of smoke detection devices may not provide sufficient advance warning for complete evacuation of the occupants. In high-rise buildings smoke generated by a fire may move throughout the building either through the air handling system or by stack effect generated due to differences in temperature between the inside and the outside of the building. In order to minimize the exposure of occupants to the toxic products of a fire in high-rise buildings, methods have been developed that use the building air handling system for the control of smoke movement within the building [96]. A typical smoke control system for a high-rise building would, in the event of a fire, place the air handling zone servicing the fire floor in a 100% exhaust mode. The air handling zones above and below the zone of fire origin are placed in a 100% supply mode such that the resulting pressure differentials will restrict the movement of smoke beyond the fire zone. The use of smoke control systems described above can maintain major portions of a high-rise building under essentially smoke free conditions and thus allow the occupants to remain in the building for the duration of the fire. It should be noted however that it is still necessary to evaluate those persons within the air handling zone where the fire originates.

6. CONCLUSIONS

The present state-of-the-art in combustion toxicology is in a state of rapid change. For a number of years it was assumed that the toxicological hazard of combustion products would be determined with a chemical analysis of selected toxic products. Due to the complexity of the product mixture and possible interaction of toxicants, this is no longer considered feasible and within the last few years an increasing number of laboratories have been using biological methods.

The predominant animal model being used to develop combustion toxicity data is the rat. The toxicological results or relative toxicity of combustion products from various materials will depend strongly on the method used to generate the products. Consequently, the relative ranking of materials between various laboratories are likely to be highly variable and appear to be inconsistent. The biological endpoint, that is, death or incapacitation, may also affect the relative ranking or toxicological hazard believed to be associated with combustion products from various materials.

Until more data and experience has been accumulated in this type of hazard assessment, it will be difficult to assess small differences in materials. However, orders of magnitude in relative toxicity can be determined using a bioassay technique.

The reader should recognize that the emphasis of this review is toxicity of combustion products and any fire hazard assessment must take into account other factors, such as ease of ignition and flame spread rate of the material in question.

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Table 1. Summary of Combustion Toxicology Systems

Method	Burning Conditions				Animal Exposure Conditions					
	Mode	Temp.	Airflow	Time (min)	Time (min)	Species	Atmosp.	Chamber Configuration	Endpoint	Measurements Analytical & Biological
FAA (P. Smith) [50]	tube furnace	600 °C	controlled (closed loop)	time to death 30	time to death 30	rats 150 - 300 gm	air 30 °C	rectangular with rotating wheel 12.6 ℓ	time to Incapacitation and death	HCN, O_2, CO_2, CO pathology
Univ. of Utah (Einhorn) [50]	radiant flux	2.5, 5 & 7.5 W/cm^2	uncontrolled	5	30	rats	air	NBS Chamber & acrylic box	death & loss of trained reflex	CO blood chemistry (pH COHb, O_2, Hb, ECC*, EEG**, etc.)
U. S. Testing (Rider) [50]	radiant 45° angle	2.5 W/cm^2	16 ℓ/min	time of first death	30 or less + 14 days	rats (albino) 180 - 250 gm	8 ℓ/min air induced 30 °C	40 ℓ glass	LD_{50}	O_2, Temp. COHb
Bayer (Kimmerle) [51]	moving tube furnace	200 - 600 °C (at sample)	100 ℓ/hr 5g	30	time to death	rats	dilute products with 100 ℓ/hr	50 ℓ glass (20 rats) nose exposed only	death	CO, HCN, T, COHb
Univ. of Paris (Jouany) [50]	moving tube furnace	400 - 500 840 °C	120 ℓ/hr	30	30	rabbits rats	dilute products with 100 ℓ/hr	flow through forced ventilation	Intoxication Index	O_2, CO_2, CO, HCl HCN. pO_2, pCO_2, pH. COHb, ECG*, EEC**
University of Michigan (Cornish) [50]	1. tube furnace	700 °C (at furnace)	uncontrolled	6 - 10	4 hrs	rats	air	1500 S.S. static	LC_{50}	CO, CHl, HCN, NO_x COHB gross pathology
	2. tube furnace	5°/min to 700 °C (at furnace)	1 ℓ/hr)	140	140	rats	2 ℓ/hr added	flow through nose exposed only	LC_{50}	
Univ. of Ghent (Herpol) [51]	moving tube furnace	400 - 800 °C (at furnace)	200 ℓ/hr	30	30	wistar rats [6]	200 ℓ/hr added	nose exposure flow through	time to death	CO, CO_2, O_2, COHb RR† pathol.

* ECG - Electrocardiogram

** EEG - Electroencephalogram

† RR - Respiratory rate

Table 1, continued

Method	Burning Conditions				Animal Expoaure Conditions					
	Mode	Temp.	Airflow	Time (min)	Time (min)	Species	Atmosp.	Chamber Configuration	Endpoint	Measurements Analytical & Biological
Harvard Univ. (Dressler) [50]	radiant unknown flux	ignition max. of 1000 °C	0-200 ℓ/h	15	15	rats	air	wheel 200	Incapacitation & death	CO, O_2, CO_2, COHB
Douglas Air Craft (Caume) [50]	full-scale	flaming	uncontrolled	30	30	mice	air	running wheel	TUF	Temperature ECC*, RR†
Univ. of Pitts. (Alarie) [50]	Thermal Balance	5 °C/min	air 7 ℓ/min	30	30	mice	dilution air	flow through nose exposure only	respiratory rate decrease of 50%	respiratory rate
Japan (Kishitani) [95]	tube furnace	350, 500 750 °C	4 ℓ/min	15	15	mice	air	56 ℓ	time to death	CO, CO_2, O_2, HCl, HCN, COHb, EEG**
RASF (Hofmann) [52]	moving tube furnace	300-600°C	5 g/100 ℓ/h	30	30	rats	dilute products with 100 air	flow through nose exposure	death	COHb
NBS - JHU Birky [50]	full-scale	combustion	uncontrolled	30	30	rats	air	wheel	death incapacitation	COHb, blood CN pathology
NASA - UN. of San Francisco (Marcussen) [50]	full-scale	combustion	uncontrolled	10	10	rats	air	wire cage	death	COHb, blood cyanide pathology
Fire Research St. (Bowes) [96]	fire propag. test	combustion	controlled	60	30 (after decomp.)	rat guinea pig	21 % O_2 restored	10 m^3 ambient	death, LC_{50}	COHb, O_2, CO, CO_2, HCl

* ECG - Electrocardiogram

** EEG - Electroencephalogram

† RR - Respiratory rate

Table 2. Effects in Rats Exposed 30 Minutes to Combinations of Hydrogen Cyanide and Carbon Monoxide [51].

Carbon Monoxide (ppm)	Hydrogen Cyanide (ppm)	Mortality (%)
3,300	107	100
3,300	57	65
3,300	45	50
3,300	27	20
3,300	14	0
4,400	53	100
4,400	44	75
4,400	38	45
4,400	30	40
4,400	21	30
4,400	12	20

Table 3. Speculations on the Level of Response Which Would Occur in Humans from Results Obtained in Mice [75]

Results Obtained in Mice	Predicted Reactions in Humans
Concentration = RD_{50}	Intolerable and rapidly incapacitating
Concentration = 1/10 RD_{50}	Slightly irritating with burning sensation of the eye-nose-throat
Concentration = 1/100 RD_{50}	Tolerable with very slight or no irritating sensation

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16. ABSTRACT (A 200-word or less factual summary of most significant information. If document includes a significant bibliography or literature survey, mention it here.)

The purpose of this effort is to review what is known about the "smoke inhalation" hazard as related to human fatalities, the limitations of the fire fatality data and the methods that have been and are being used to assess the inhalation toxicity hazard.

Fire statistics indicate that 70 to 80% of the fire fatalities are attributed to smoke inhalation. In depth autopsy studies of some of these fatalities show that carbon monoxide is the predominant toxicant produced from fires. The role of new synthetic polymers and other additives is unknown as is the role of hydrogen cyanide in fire fatalities.

Chemical analysis of combustion products has been used extensively to assess the toxicological hazard in fire research. The limitations of such measurements are addressed and a combination of toxicology (animal exposures) and analytical chemistry is recommended. Recent combined biological and selected analytical measurements are critically reviewed.

The mechanism of toxic action of a few well known combustion products is discussed. Due to the extensive use of organophosphates as fire retardants in polymeric materials, the toxicity of this class of compounds is reviewed in some detail.

The role of building codes and standards and early detection and suppression of fire are discussed as a means of reducing human exposure to toxic combustion products. For example, the 1976 French regulation that limits the use of flammable materials that contain chlorine and nitrogen in their molecular structure is presented.

17. KEY WORDS (six to twelve entries; alphabetical order; capitalize only the first letter of the first key word unless a proper name; separated by semicolons)

Biological assessment; chemical analysis; combustion products; fire fatalities; smoke inhalation; toxicity.

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